DOI: 10.16899/ctd.33991
OriginalArticle / Orijinal Araştırma

Düşük Doz Topiramat Kullanımı Sonrası Gelişen Bilateral Akut Dar Açılı Glokom ve Miyopi

Bilateral Acute Angle Closure Glaucoma and Myopia Induced by Low Dosage Topiramate

Büşra S. Arıca[1], Emrullah Beyazyıldız[2], Özlem Beyazyıldız[3]

[1]Gazi Mustafa Kemal State Hospital, Department of Neurology.

[2]Gazi Mustafa Kemal State Hospital, Department of Ophthalmology.

[3]Ulucanlar Eye Research and Training Hospital, Department of Ophthalmology

Corresponding Author:

Dr.Büşra S. Arıca

Address:
Gazi Mustafa Kemal State Hospital, Department of Neurology.
Gazi Mah. Silahtar cad. No: 2 Yenimahalle/ANKARA-TURKEY.

Tel: 90 312 212 66 66

E-mail:
busrarica@yahoo.com

Başvuru Tarihi/Received :

08-01-2014

Kabul Tarihi/Accepted:

28-02-2014

ÖZET

Giriş: Topiramat, epilepsi tedavisinde ve migren profilaksisinde etkili olduğu gösterilen sülfamat sübstitüsyonlu monosakkarid türevi bir ajandır. Bu ilacın kullanımı sırasında özellikle ilk iki hafta içerisinde akut myopi ile birlikte açı kapanması glokomu krizi gelişebilir.

Olgu: Bu olgu sunumunda aurasız migren profilaksisi nedeniyle bir haftadır topiramat 25 mg/gün kullanımı sonrası açı kapanması glokomu ve akut myopi gelişen 23 yaşında bayan hasta tartışılmıştır. Görme kaybı, gözlerde kızarıklık ve yanma şikayetiyle başvuran hastanın yapılan oftalmolojik muayenesinde her iki gözde belirgin konjonktival hiperemi, sığ ön kamara, iriste bombeleşme ve her iki gözde grade 1 dar açı saptandı. Intraoküler basınç her iki gözde 40 mmHg idi. Her iki gözde refraksiyon değerleri -7,00 ve -8,00 olarak saptandı. Hastanın görme keskinliği seviyeleri 0,1 ile 0,2 düzeyindeydi. Diğer nörolojik muayene bulguları normal olarak değerlendirildi. Hastanın almakta olduğu topiramat tedavisi kesildi ve topikal antiglokomatöz ilaç tedavisi başlandı ve her iki göze periferal iridotomi yapıldı. On gün sonraki oftalmolojik muayenesi tamamen normaldi.

Yorum: Topiramat kullanımına bağlı bu yan etkinin erken saptanması sonrasında ilacın kesilmesi ve antiglokomatöz tedavi ile , oluşan tüm bulguların hasar bırakmaksızın ortadan kalktığı bilinmektedir. Bu yüzden özellikle topiramat tedavisi başlandıktan sonraki ilk haftalarda hastaların ve hekimlerin göz bulguları açısından dikkatli olmaları gerekmektedir.

Anahtar sözcükler: Glokom, migren, miyopi, topiramat

ABSTRACT

Introduction: Topiramate, a sulfamate-substituted monosaccharide, has been shown to be effective in the treatment of epilepsy and migraine prophylaxis. However, acute secondary angle closure glaucoma and myopia has been shown to develop, especially during the first two weeks of treatment, in a small subset of patients.

Case presentation: In the current case report, a 23 year old female patient developed acute myopia and angle closure glaucoma after one week topiramate treatment (25 mg/day) for prophylaxis of migraine without aura. The patient was found to have significant conjunctival hyperemia, shallow anterior chamber, and bulging iris in both eyes. Grade 1 acute angle was detected in both eyes during gonioscopic examination. There was no pupillary block and intraocular pressure was 40 mmHg in both eyes. Refraction values were measured at -7.00 and -8.00 in the right and left eye, respectively. The patient's visual acuity was at 0.1 to 0.2.

Topiramate treatment was promptly discontinued, topical antiglaucomatous treatment was initiated, and laser peripheral iridotomy was performed on each eye. Intraocular pressure has declined to normal limits, refractive values were zero in both eyes and patient's visual acuity has restored at follow-up period at 10 days after treatment.

Conclusion: Side effects associated with topiramate treatments are known to disappear without long-term damage when the discontinuation of therapy and effective interventions are started early. Therefore, patients and their physicians should be alert for symptoms associated with acute secondary angle closure glaucoma and myopia; especially in the first weeks of topiramate treatment also with low dosage.

Key words: Glaucoma, migraine, myopia, topiramate

INTRODUCTION

Topiramate, which is known to be effective in the treatment of epilepsy and migraine prophylaxis, is a sulfamate-substituted monosaccharide derivative related to fructose (1,2). Common side effects associated with topiramate treatment include fatigue, paresthesia, taste disturbance, weight loss and imbalance. Several publications have reported the development of ocular side effects which in rare cases have led to serious morbidity of the eye tissue (3,4). In addition, acute angle-closure glaucoma and myopia has been shown to develop with topiramate therapy particularly during the first two weeks of treatment. Several mechanisms underlying these side effects have been proposed, but there is still no clear consensus in the field (4). Early detection and treatment is very important that prevent permanent vision loss as a result of ocular side effects. In the current study, we aimed to discuss a particular case that was admitted to our clinic with angle-closure glaucoma and acute myopia statement following one week topiramate treatment (25 mg/day).

CASE REPORT

The 23 year old female patient, on topiramate therapy for migraine (without aura) prophylaxis treatment for one week, was admitted to the outpatient clinic with complaints of headache, loss of vision, red eye, and tearing that had persisted for three days. The patient did not have a family history of glaucoma, did not wear glasses, and had no prior visual problems.

Consciousness, orientation and cooperation were all found to be normal with a neurological examination. There were no signs of meningeal irritation, but conjunctival hyperemia was present in both eyes (see below). Pupils were at midline, isochoric, and light reflexes preserved. The optical disc had a natural appearance, nystagmus was absent, and eye movements were normal in all directions. Other cranial nerve examinations were within normal limits, and there was no apparent loss of muscular strength. Both cerebellar and sensory examinations were normal. Lastly, deep tendon reflexes were within the normal range and no pathological reflexes were observed. At this point, the patient was referred to an ophthalmologist for further evaluation.

From the ophthalmic examination, the patient was found to have significant conjunctival hyperemia, shallow anterior chamber, and bulging iris in both eyes. Grade 1 acute angle was detected in both eyes during gonioscopic examination. There was no pupillary block and lenticular contact with the iris, and intraocular pressure was 40 mmHg in both eyes. Importantly, the patient's corneas were clear. Refraction values were measured at -7.00 and -8.00 in the right and left eye, respectively. The patient's best corrected visual acuity was at 0.1 to 0.2.

Based on the symptoms presented by the patient, topiramate treatment was discontinued. In addition, topical anti-glaucoma treatment (bromonidine + dorzolamide-timolol) was initiated and peripheral iridotomy was performed on both eyes. At the follow-up ophthalmological examination (10 days post-treatment), intraocular pressure was in normal limits (10 and 12 mmHg, respectively), the patient's visual acuity was equal and 1.0 in both eyes, conjunctival hyperemia was no longer present, refractive values were zero in both eyes, and corneal edema no longer observed. Gonioscopic examination revealed that iridocorneal angle was open and grade 3 in both eyes.

DISCUSSION

In this case study, one week topiramate treatment led to the development of acute myopia and glaucoma in a 23 year old female patient. Topiramate, which is commonly prescribed as an antiepileptic, can also be used for prophylactic treatment of a wide range of disorders including: migraine, depression, bipolar disorder, posttraumatic stress disorder, neuropathic pain, and postherpetic neuralgia (2). Clinical signs of ocular side effects due to

topiramatetherapy can develop rapidly, and may include: bilateral blurred vision, eye redness and tearing, nausea, vomiting and headache. These side effects are most common in the first few weeks of treatment with topiramate. Upon ophthalmological examination, conjunctival hyperemia, corneal edema, anterior chamber inflammation, and increased intraocular pressure are observed. These side effects occur in ~3% of patients receiving tomiramate therapy. Diagnoses are generally made by clinical observation (of the symptoms described above) and confirmed with the detection of uveal effusion using ultrasound biomicroscopy (5).

In a recent study which performed on patients receiving topiramate therapy, 86 of 115 developed glaucoma and 17 of 115 developed bilateral acute myopia (4). To date, the mechanisms underlying the ocular side effects associated with topiramate treatment have not be determined (6). However, some have suggested that these side effects are the result of ciliary body edema and anterior displacement of the ciliary processes (7). In some patients, the ciliary body forward displacement was shown using ultrasound biomicroscopy (8,9). It is also possible that the myopic shift observed in patients with uveal effusion and edema of the ciliary body will result in the forward displacement of the lens-iris diaphragm and shallowness of anterior chamber (6,10,11). Saffra*et. al.* observed uveal effusion in patients with advanced glaucoma and refractive change following topiramate treatment, but found that topical anti-glaucoma medications decreased uveal effusion, improved refractive changes and decreased the eye tension to normal levels (8).

It is believed that the primary cause of blurred vision in patients during topiramate treatment is acute myopic shift (12). However, corneal edema may also develop and could also contribute to blurred vision in these patients (13). In the case study reported here, myopic shift was enhanced to -7.00 while corneal edema was absent, suggesting that myopic shift was sufficient in this patient to lead to significant loss of vision. The lack of corneal edema observed (which is commonly observed in other case studies) is likely the result of the age of the patient. In addition to the discontinuation of topiramate therapy, our patient was treated with topical anti-glaucoma medications (dorzolamide + timolol with bromonidine) and laser iridotomy was performed. The benefit of laser iridotomy in cases with a lack of pupillary block has not been demonstrated, but there is a consensus that discontinuation of topiramate therapy, antiglaucoma medical treatment and the use of steroids can effectively treat common ocular side effects (4,5,14, 15). The patient did not respond adequately to treatment with laser iridotomy, and effective results were only observed after antiglaucomatous treatment.

Based on our findings and those of others, we propose that all patients should receive an ophthalmological examination to check for the presence of narrow angle and should not have a family history of glaucoma before starting topiramate therapy. Importantly, with early diagnosis and effective treatment after discontinuation of topiramate, all observed side-effects disappear without leaving any long-term damage (15). Therefore, patients and their physicians should be alert for these ocular side effects, especially in the first weeks, after the initiation of topiramate treatment also with low dosage.

REFERENCES

1. Maryanoff BE. Sugarsulfamatesforseizurecontrol: discoveryanddevelopment of topiramate, a structurallyuniqueantiepilepticdrug. CurrentTopics in MedicinalChemistry. 2009;9(11):1049–1062
2. JohannessenLandmark C. Antiepilepticdrugs in non-epilepsydisorders: relationsbetweenmechanisms of actionandclinicalefficacy. CNS Drugs. 2008;22(1):27–47.
3. Spaccapelo L, Leschiutta S, Aurea C, Ferrari A. Topiramate-associatedacuteglaucoma in a migrainepatientreceivingconcomitantcitalopramtherapy: a case-report. CasesJournal 2009,2:87
4. Fraunfelder FW, Fraunfelder FT, Keates EU. Topiramate-associatedacute, bilateral, secondaryangle-closureglaucoma.Ophthalmology. 2004 Jan;111(1):109-11.
5. Panday VA, RheeDJ.Review of sulfonamide-inducedacutemyopiaandacutebilateralangle-closureglaucoma.ComprOphthalmol Update. 2007 Sep-Oct;8(5):271-6.
6. Craig JE, Ong TJ, Louis DL, Wells JM. Mechanism of topiramate-inducedacute-onsetmyopiaandangleclosureglaucoma.Am J Ophthalmol. 2004 Jan;137(1):193-5.

7. Banta JT, Hoffman K, Budenz DL, Ceballos E, Greenfield DS. Presumedtopiramateinducedbilateralacuteangleclosureglaucoma. Am J Ophtalmol.2001;132:112-114

8. Saffra N, Smith SN, SeidmanCJ.Topiramate-inducedrefractivechangeandangleclosureglaucomaanditsultrasoundbi microscopyfindings. BMJ Case Rep. 2012 Aug 1;2012

9. Medeiros FA, Zhang XY, Bernd AS, Weinreb RN. Angle-closureglaucomaassociatedwithciliary body detachment in patientsusingtopiramate. Arch Ophthalmol.2003;121:282-284

10. Paciuc-Beja M, Retchkiman-Bret M, Velasco-Barona CF, Galicia-Alfaro VH. SecondaryBilateralAngleClosureGlaucomaduetoTopiramate.CaseRep OphthalmolMed. 2011;2011

11. Sankar PS, Pasquale LR, Grosskreutz CL. Uvealeffusionandsecondaryangle-closureglaucomaassociatedwithtopiramateuse. Arch Ophthalmol.2001;119:1210-1211.

12. Örüm Ö, Tarakçıoğlu H, Yiğit U. Topiramat kullanımının tetiklediği miyopik kayma ve bilateralsekonder kapalı açılı glokom. Turk J Ophthalmol 2012;42:154-6

13. Willett MC, Edward DP. Refractorytopiramate-inducedangle-closureglaucoma in a man: a casereport. J Med Case Reports.2011;26-33

14. Sbeity Z, Gvozdyuk N, Amde W, Liebmann JM, Ritch R. Argon laserperipheraliridoplastyfortopiramate-indusedbilateralacuteangleclosure. J Glaucoma.2009;18:269-71

15. Lorenzatti M, Nazzarro V, Abdolrahimzadeh S. Bilateralacuteangleclosureglaucomaandmyopiainducedbytopiramate.C lin Ter. 2009;160(3):215-6